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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/729,427	12/05/2003	Kevin J. Tracey	3268.1005-001	8405

21005 7590 12/21/2005

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EXAMINER	
GRAFFEO, MICHEL	
ART UNIT	**PAPER NUMBER**
1614	

DATE MAILED: 12/21/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/729,427	TRACEY ET AL.	
	Examiner	Art Unit	
	Michel Graffeo	1614	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *09 November 2005*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1,4,10-15 and 20-23* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1, 4, 10-15 and 20-23* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Status of Action

Claims 1, 4, 10-15 and 20-23 are pending and examined.

Applicant has amended claim 1, canceled claims 2-3, and provided arguments for the patentability of claims 1, 4, 10-15 and 20-23 in the response filed 9 November 2005. Any rejection not specifically stated in this Office Action has been withdrawn.

Response to Election/Restrictions

Applicant's election with traverse of Group I noted in the Office Action dated 8 June 2005 is acknowledged. No grounds for the traversal were given and the requirement is deemed proper and is therefore made FINAL.

Maintained Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation

under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1-4, 10-15 and 20-23 are rejected under 35 U.S.C. 103(a) as being unpatentable over Borovikova et al. Vagus nerve stimulation attenuates the systemic inflammatory response to endotoxin. Letter to Nature 2000; 405:458-462 in view of Moreland et al. Treatment of Rheumatoid Arthritis with a Recombinant Human Tumor Necrosis Factor Receptor (p75)-Fc Fusion Protein. The New England Journal of Medicine 1997:337: 141-147 and further in view of US Patent Number 5,977,144 to Meyer et al.

Borovikova et al. teach that the α-bungarotoxin-sensitive nicotinic acetylcholine receptors (see Meyer et al. below which teach that the α-7 nicotinic receptors were in fact α-bungarotoxin-sensitive nicotinic acetylcholine receptors) are required for inhibition of tumor necrosis factor (TNF) and that these receptors mediate the TNF response in macrophages (see page 459 second column and page 461 first column).

Borovikova et al. do not teach that an anabaseine derivative can be used to agonize the receptor and subsequently treat rheumatoid arthritis (RA).

Moreland et al. teach that TNF plays a role in the pathogenesis of RA (see page 141 col 2) and suggests that a reduction of TNF may reduce the activity of RA (see Abstract background).

Meyer et al. teach that anabaseine derivatives and in particular that 3-(4-hydroxy-2-methoxybenzylidene) anabaseine (see col 5 line 4) and 3-(2,4-dimethoxybenzylidene) anabaseine (see col 1 line 46) selectively target the α-7 nicotinic receptor (see col 3 lines 48-58 and the Abstract) and that the α-7 nicotinic receptors are in fact α-bungarotoxin-sensitive nicotinic acetylcholine receptors (see col 1 lines 38-45).

One skilled in the art is motivated to combine the above references. The advantages of each reference as combined is directed to the teaching of treating RA with an anabaseine compound via the modulation of TNF. Specifically, Moreland et al. teach that modulation of TNF can treat RA and that interference with the cytokine cascade may be of additional benefit in treating RA (see page 146 2nd column). Therefore, one skilled in the art would be motivated to look to the mechanisms by which one can modulated TNF. Borovikova et al. teach that TNF synthesis can be inhibited (see page 458 col 2) by direct stimulation of the vagus nerve, which is analogous to an increase of acetylcholine. Borovikova et al. also teach that a decrease in TNF is further known to be effected via the α-bungarotoxin-sensitive nicotinic acetylcholine receptor pathway. Moreover both Moreland et al. and Borovikova et al. address the issue of inflammation (see col 2 in Moreland and page 459 col 2 in Borovikova et al.), a common symptom of RA, and the role of TNF therein. One skilled in the art would be further motivated to combine Meyer et al. with Borovikova et al. since one skilled in the art looking to agonize the α-7 receptor would be motivated to look to popular and known α-7 receptor agonists such as those described in Meyer et al. Thus, the claimed invention

of the composition was within the ordinary skill in the art to make and use at the time it was made and was as a whole, *prima facie* obvious.

Response to Arguments

Applicant's arguments filed 9 November 2005 have been fully considered but they are not persuasive. In response to applicant's argument that the examiner's conclusion of obviousness is based upon improper hindsight reasoning, it must be recognized that any judgment on obviousness is in a sense necessarily a reconstruction based upon hindsight reasoning. But so long as it takes into account only knowledge which was within the level of ordinary skill at the time the claimed invention was made, and does not include knowledge gleaned only from the applicant's disclosure, such a reconstruction is proper. See *In re McLaughlin*, 443 F.2d 1392, 170 USPQ 209 (CCPA 1971).

Meyer et al. provides the motivation and knowledge within the level of ordinary skill at the time the claimed invention was made. To that end, Meyer et al. teaches that the α-7 receptor demonstrates a characteristic high affinity binding to α-bungarotoxin, high calcium-permeability and rapid desensitization (see col 1 lines 39-42) and goes on further to teach that DMXB is the best studied selective α-7 agonist (see col 1 lines 45-47). Meyer et al. combined with Borovikova et al. which teaches that TNF is modulated in response to all α-bungarotoxin receptors on page 459 which states in part that "...TNF response in human macrophage cultures is mediated primarily by α-

bungarotoxin..." receptors. Therefore, one of ordinary skill in the art, absent evidence to the contrary, would have been motivated to use DMXB to modulate TNF and knowingly treat RA.

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. See *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970);and, *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent is shown to be commonly owned with this application. See 37 CFR 1.130(b).

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

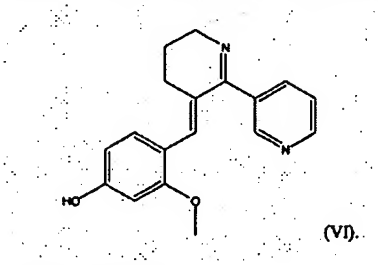
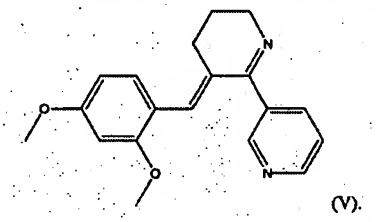
Claims 1-4, 10-15 and 20-23 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-8, 11-14 and 18 of U.S. Patent No. 6,838,471 to Tracey in view of US Patent No. 5,977,144 to Meyer et al. since the claims, if allowed, would improperly extend the "right to exclude" already granted in the patent.

The subject matter claimed in the instant application is fully disclosed in the patent and is covered by the patent since the patent and the application are claiming common subject matter, as follows in the comparison table below:

Claims of 10/729427	Claim limitations of '427	Claim limitations in US 6,838,471 reference

1	A method of treating a patient with an anabaseine derivative selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory cytokine that is released from a macrophage wherein the condition is RA.	Claim 5: A method for inhibiting the release of a proinflammatory cytokine from a cell in a patient suffering from a condition mediated by an inflammatory cytokine comprising treating the cell with an agonist* selective for the α-bungarotoxin-sensitive nicotinic acetylcholine receptor to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See Meyer et al. at col 1 lines 35-42 which teaches that the α-bungarotoxin-sensitive nicotinic acetylcholine receptor is the α-7 nicotinic receptor. See also claims 1-4, 6-8, 11-14 and 18.
2	A method of treating a patient with an anabaseine derivative selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory cytokine, such as TNF for example, that is released from a macrophage wherein the condition is RA.	Claim 3: A method for inhibiting the release of TNF from a cell to the extent that cell is in a patient, comprising treating the cell with an agonist selective for the α-bungarotoxin-sensitive nicotinic acetylcholine receptor to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1, 2, 4-8 and 11-14 and 18.
3	A method of treating a patient with an anabaseine derivative selective for an α-7 nicotinic receptor to decrease the amount of TNF that is released from a macrophage wherein the condition is RA.	Claim 3: A method for inhibiting the release of TNF from a cell to the extent that cell is in a patient, comprising treating the cell with an agonist selective for the α-bungarotoxin-sensitive nicotinic acetylcholine receptor to decrease the amount of proinflammatory cytokine

		wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1, 5-8 and 11-14 and 18.
4	Same as claim 1	
10	Same as claim 1	
11	A method of treating a patient with certain anabaseine derivatives selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory cytokine that is released from a macrophage wherein the condition is RA.	Claim 5: A method for inhibiting the release of a proinflammatory cytokine from a cell in a patient suffering from a condition mediated by an inflammatory cytokine comprising treating the cell with an agonist* selective for the α-bungarotoxin-sensitive nicotinic acetylcholine receptor to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1-4, 6-8, 11-14 and 18
12	A method of treating a patient with certain anabaseine derivatives selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory cytokine that is released from a macrophage wherein the condition is RA.	Claim 5: A method for inhibiting the release of a proinflammatory cytokine from a cell in a patient suffering from a condition mediated by an inflammatory cytokine comprising treating the cell with an agonist* selective for the α-bungarotoxin-sensitive nicotinic acetylcholine receptor to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1-4, 6-8, 11-14 and 18
13	A method of treating a patient with certain anabaseine derivatives selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory	Claim 5: A method for inhibiting the release of a proinflammatory cytokine from a cell in a patient suffering from a condition mediated by an inflammatory

	cytokine that is released from a macrophage wherein the condition is RA.	cytokine comprising treating the cell with an agonist* selective for the α-bungarotoxin-sensitive nicotinic acetylcholine receptor to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1-4, 6-8, 11-14 and 18
14	A method of treating a patient with the anabasein derivative:  (VI). selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory cytokine that is released from a macrophage wherein the condition is RA.	Claim 5: A method for inhibiting the release of a proinflammatory cytokine from a cell in a patient suffering from a condition mediated by an inflammatory cytokine comprising treating the cell with an agonist* selective for the α-bungarotoxin-sensitive nicotinic acetylcholine receptor to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1-4, 6-8, 11-14 and 18
15	A method of treating a patient with the anabaseine derivative:  (V). selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory cytokine that is released from a macrophage wherein the condition is RA.	Claim 5: A method for inhibiting the release of a proinflammatory cytokine from a cell in a patient suffering from a condition mediated by an inflammatory cytokine comprising treating the cell with an agonist* selective for the α-bungarotoxin-sensitive nicotinic acetylcholine receptor to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1-4, 6-8, 11-14 and 18
20	Same as Claim 1	

21	Same as Claim 1	
22	Same as Claim 1	
23	Same as Claim 1	

* See US 5977144 to Meyer et al. which teaches anabaseine derivatives as α-7 nicotinic receptor agonists. Particular anabaseine derivatives include those of claims 13 and 14 above. It would be obvious to one skilled in the art to combine Meyer et al. with the '471 reference. Both are directed to the targeting of the α-7 receptors and one skilled in the art would find it obvious to use the well studied agonists of Meyer et al.

Claims 1-4, 10-15 and 20-23 are rejected under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1-3, 5-6, 14 and 16 of U.S. Patent No. 6,610,713 to Tracey in view of US Patent No. 5,977,144 to Meyer et al. since the claims, if allowed, would improperly extend the "right to exclude" already granted in the patent.

The subject matter claimed in the instant application is fully disclosed in the patent and is covered by the patent since the patent and the application are claiming common subject matter, as follows in the comparison table below:

Claims of 10/729427	Claim limitations of '427	Claim limitations in US 6,610,713 reference
1	A method of treating a patient with an anabaseine derivative selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory cytokine that is released from a macrophage wherein the condition is RA.	Claim 5: A method for inhibiting the release of a proinflammatory cytokine from a cell in a patient suffering from a condition mediated by an inflammatory cytokine comprising treating the cell with a cholinergic agonist* to decrease the amount of proinflammatory cytokine

		wherein the cell is a macrophage and to the extent that the condition is RA. See also claims 1-3, 6, 14 and 16.
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3	A method of treating a patient with an anabaseine derivative selective for an α-7 nicotinic receptor to decrease the amount of TNF that is released from a macrophage wherein the condition is RA.	Claim 3: A method for inhibiting the release of TNF from a cell to the extent that cell is in a patient, comprising treating the cell with a cholinergic agonist to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1, 5, 6, 14 and 16.
4	Same as claim 1	
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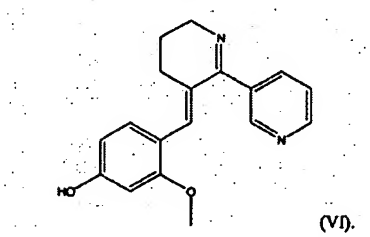
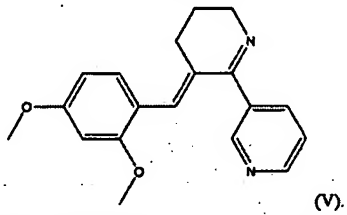
14	A method of treating a patient with the anabasein derivative:  (VI). selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory cytokine that is released from a macrophage wherein the condition is RA.	Claim 5: A method for inhibiting the release of a proinflammatory cytokine from a cell in a patient suffering from a condition mediated by an inflammatory cytokine comprising treating the cell with a cholinergic agonist* to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1-3, 6, 14 and 16.

15	A method of treating a patient with the anabaseine derivative:  (V). selective for an α-7 nicotinic receptor to decrease the amount of proinflammatory cytokine that is released from a macrophage wherein the condition is RA.	Claim 5: A method for inhibiting the release of a proinflammatory cytokine from a cell in a patient suffering from a condition mediated by an inflammatory cytokine comprising treating the cell with a cholinergic agonist* to decrease the amount of proinflammatory cytokine wherein the cell is a macrophage to the extent that the condition is RA. See also claims 1-3, 6, 14 and 16.
20	Same as Claim 1	
21	Same as Claim 1	
22	Same as Claim 1	
23	Same as Claim 1	

* See US 5977144 to Meyer et al. which teaches the cholinergic agonist anabaseine derivatives as α-7 nicotinic receptor agonists. Particular anabaseine derivatives include those of claims 13 and 14 above. It would be obvious to one skilled in the art to combine Meyer et al. with the '471 reference. Both are directed to the targeting of the α-7 receptors and one skilled in the art would find it obvious to use the well studied agonists of Meyer et al.

Response to Arguments

Applicant's arguments filed 9 November 2005 have been fully considered but they are not persuasive. In response to applicant's argument that the examiner's conclusion of obviousness is based upon improper hindsight reasoning, it must be recognized that any judgment on obviousness is in a sense necessarily a reconstruction

based upon hindsight reasoning. But so long as it takes into account only knowledge which was within the level of ordinary skill at the time the claimed invention was made, and does not include knowledge gleaned only from the applicant's disclosure, such a reconstruction is proper. See *In re McLaughlin*, 443 F.2d 1392, 170 USPQ 209 (CCPA 1971).

As noted above, Meyer et al. provides the motivation and knowledge within the level of ordinary skill at the time the claimed invention was made. To that end, Meyer et al. teaches that the α-7 receptor demonstrates a characteristic high affinity binding to α-bungarotoxin, high calcium-permeability and rapid desensitization (see col 1 lines 39-42) and goes on further to teach that DMXB is the best studied selective α-7 agonist (see col 1 lines 45-47). Meyer et al. combined with Borovikova et al. which teaches that TNF is modulated in response to all α-bungarotoxin receptors on page 459 which states in part that "...TNF response in human macrophage cultures is mediated primarily by α-bungarotoxin..." receptors. Therefore, one of ordinary skill in the art, absent evidence to the contrary, would have been motivated to use DMXB to modulate TNF and knowingly treat RA. In addition, applicant's argument in the response does not demonstrate how these claims are not obvious variations.

Conclusion

No claim is allowed.

THIS ACTION IS MADE FINAL. Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Examiner Graffeo whose telephone number is 571-272-8505. The examiner can normally be reached on 9am to 5:30pm Monday to Friday.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christopher Low can be reached on 571-272-0951. The fax phone number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

12 December 2005
MG

MG

Christopher S.F. Low
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